Developmental Biology 331 (2009) 507–516

Contents lists available at ScienceDirect

Developmental Biology

journal homepage: www.elsevier.com/developmentalbiology

Abstracts

Cell fate specification

Program/Abstract # 420
Control of cell and tissue polarity in neurectoderm by the PAR-1 (MARK) proteins in *Xenopus*

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PAR-1 (MARK) polarity regulating kinases have diverse substrates in invertebrate and vertebrate species but consistently act as polarity regulators during development. We have shown by gain- and loss-of-function studies in *Xenopus* that PAR-1 interacts with canonical and non-canonical Wnt signaling to control primary body axis polarity and shape and neuronal and ciliated cell fate specification. We present new data indicating that PAR-1 acts as a layer-identity specification factor in the ectoderm, implicating asymmetrical cell division events in primary neurogenesis. We also define the onset of secondary neurogenesis and establish the role of PAR-1 in regulating cell fate at these later stages, including effects on proliferation, differentiation and tissue architecture.

doi:10.1016/j.ydbio.2009.05.447

Program/Abstract # 421
Maintenance of progenitor fate at the neural plate border by zebrafish prdm1a

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Neural crest cells and Rohon-Beard (RB) sensory neurons are induced at the neural plate border, but the molecular mechanisms that are required for their induction remain unclear. We hypothesize that multiple different progenitors exists for both neural crest and RB sensory neurons and that similar molecular mechanisms are required for their induction. Analysis of the prdm1a mutation suggests a genetic link between trunk neural crest and RB sensory neurons since both cell types are affected in the mutation, where RB neurons are completely absent and neural crest cells and their derivatives are reduced. Further analysis of gene expression at the neural plate border suggests that there is an extensive overlap between prdm1a and the epidermal marker, dlx3b, but not with neural markers sox3 or sox19a. Additionally, epistatic analysis suggests that prdm1a is upstream of dlx3b/dlx4b and neurog1(ngn1) suggesting that prdm1a acts primarily at the neural plate border by regulating transcription of neighboring domains, downstream of BMP signaling and upstream of Notch. The mechanism by which prdm1a regulates cell fate at the neural plate border is not by affecting cell proliferation or cell death but by regulating the progenitor fate such that in the absence of prdm1a, the olig4 interneuron domain is expanded. When both prdm1a and olig4 are absent, there is a slight upregulation of neural crest and RB neurons. These data in combination indicate that RB sensory neurons and neural crest cells are induced by the same embryologic and molecular mechanisms at the neural plate border, both requiring prdm1 to maintain progenitor fate.

HD05698 to KA.

doi:10.1016/j.ydbio.2009.05.448

Program/Abstract # 422
Wnt antagonism of Shh facilitates midbrain floor plate neurogenesis

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The floor plate has long been recognized as a distinct group of cells situated at the ventral midline in the vertebrate embryonic central nervous system. It is defined by the expression of Shh, a morphogen that patterns the ventral neural tube. Traditionally, the floor plate was believed to be composed of ependymal cells and, unlike the vast majority of cells in the neural tube, floor plate cells were thought to be unable to differentiate into neurons. Recently, the view of the differentiation potential of the floor plate has changed dramatically. Genetic and *in vitro* lineage analyses demonstrate unequivocally that *Shh*+ midbrain floor plate cells give rise to midbrain dopamine neurons. As yet, little is known about the mechanisms, which regulate whether or not the floor plate will differentiate into neurons. Distinct spatiotemporal *Shh* and *Wnt* expressions may distinguish the neurogenetic potential of these structures. Here, we reveal an inhibitory role for Shh removal of *Shh* results in neurogenesis from the hindbrain midline; conversely, high doses of Shh inhibit proliferation and dopamine neuron production in midbrain cultures. We then show that Wnt/beta-catenin signaling is necessary and sufficient for antagonizing *Shh*, dopamine progenitor marker induction, and promotion of dopaminergic neurogenesis. These studies demonstrate how the dynamic interplay of canonical